

the incidence of high blood pressure in their patient population.

[This letter was received from a patient who asked not to be identified—Ed.]

Treatment of enuresis

I am disappointed that Netley and colleagues (*Can Med Assoc J* 1984; 131: 577–579) make no mention of Azrin and Besalel's Dry Bed Program¹ in their article on primary enuresis. Azrin and Besalel's guide, originally published in 1974, outlines an overlearning method for the treatment of enuresis. When this method is used in conjunction with a waking device, the success rate in children aged 3 to 4 years and older is as high as 95%.

In the Yukon the program is taught through the Mental Health Unit and consists of one interview with both the parent and child and then a follow-up visit approximately 3 weeks later.

The use of either imipramine hydrochloride or a waking device alone, without the behavioural techniques of overlearning, for enuresis treatment is still in the dark ages.

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Reference

1. Azrin NH, Besalel VA: *A Parent's Guide to Bedwetting Control: a Step by Step Method*, PB, New York, 1981

Ascorbic acid and kidney stones

Over the past 10 years the idea that ingestion of ascorbic acid in large doses causes kidney stones has become established. By constant repetition this idea, based entirely on conjecture, has become enshrined as a fact. The same thing happened with the erroneous theory that ascorbic acid destroys vitamin B₁₂.¹⁻³

The belief that large doses of ascorbic acid can cause kidney stones arose following a few reports that ascorbic acid was partially me-

tabolized to oxalate and that megadoses of ascorbic acid caused increases in serum and urine oxalate levels.⁴⁻⁷ Briggs and colleagues⁸ reported a man who had a tendency to produce excessive amounts of oxalate; they postulated that the risk of forming oxalate stones might be increased if he took large doses of ascorbic acid. While these papers provided the foundation for the common belief that ascorbic acid might cause kidney stones, no such event has been reported, even though millions of people take substantial doses of ascorbic acid each day.⁹

Now it turns out that the apparent increase in the urinary excretion of oxalate was due to the laboratory method used for measuring oxalate. According to Fituri and colleagues¹⁰ ingestion of 8 g of ascorbic acid daily for 7 days had no effect on serum levels or urinary excretion of oxalate.

Those who did find an increase in urine oxalate levels used a method that involved heating urine for 30 minutes at 100°C.^{4,5} But Fituri and colleagues used a method that did not require heat and observed no significant increase in urine oxalate levels; they also found that heating urine that contained ascorbic acid caused the conversion of ascorbic acid to oxalate. They concluded: "The increases found by [two previous authors] would seem to have been due to in vitro conversion of ascorbate to oxalate during the assay procedure, rather than any increased in vivo production of oxalate from the vitamin."

Yet the myth that ascorbic acid may cause kidney stones persists. In 1984, months after Fituri and colleagues' paper appeared, Alhadeff and associates¹¹ cautioned that excessive (not defined) intake of vitamin C may be associated with the formation of oxalate stones. Of the six accompanying references not one is to an original paper: all are textbooks that usually refer to others' views. Soon we will have a massive bibliography of references to authors referring to one another about a theory unsupported by experimental evidence.

There were two ideas that pointed to a connection between ascorbic acid and kidney stones: that inges-

tion of ascorbic acid would increase the serum and urine oxalate levels and that this would cause increased formation of oxalate kidney stones. The first is proven to have no basis, and the second has not occurred. Will the ascorbic acid-kidney stone myth be put to rest? I doubt it.

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References

1. Herbert V, Jacob E: Destruction of vitamin B-12 by ascorbic acid. *JAMA* 1974; 230: 241–242
2. Newmark HL, Scheiner J, Marcus M et al: Stability of vitamin B-12 in the presence of ascorbic acid. *Am J Clin Nutr* 1976; 29: 645–649
3. Marcus M, Prabhudesai M, Wassef S: Stability of vitamin B-12 in the presence of ascorbic acid in food and serum: restoration by cyanide and apparent loss. *Am J Clin Nutr* 1980; 33: 137–143
4. Lamden MP, Chrystowski GA: Urinary oxalate excretion by man following ascorbic acid ingestion. *Proc Soc Exp Biol Med* 1954; 85: 190–192
5. Takenouchi K, Aso K, Ichikawa H et al: On the metabolites of ascorbic acid, especially oxalic acid, eliminated in the urine following the administration of large amounts of ascorbic acid. *J Vitamin (Kyoto)* 1966; 13: 49–58
6. Tiselius HG, Almgard LE: The diurnal urinary excretion of oxalate and the effect of pyridoxine and ascorbate on oxalate excretion. *Eur Urol* 1977; 3: 41–46
7. Hughes C, Dutton S, Truswell AS: High intakes of ascorbic acid and urinary oxalate. *J Hum Nutr* 1981; 35: 274–280
8. Briggs MH, Garcia-Webb P, Davies P: Urinary oxalate and vitamin-C supplements [C]. *Lancet* 1973; 2: 201
9. Barness LA: Some toxic effects of vitamin C. In Hanck A, Ritzel G (eds): *Re-evaluation of Vitamin C*, Verlag Hans Huber, Bern, 1977: 19–29
10. Fituri N, Allawi N, Bentley M et al: Urinary and plasma oxalate during ingestion of pure ascorbic acid: a re-evaluation. *Eur Urol* 1983; 9: 312–315
11. Alhadeff L, Gualtieri T, Lipton M: Toxic effects of water-soluble vitamins. *Nutr Rev* 1984; 42: 33–40

Reversible captopril-associated bone marrow aplasia

Captopril, an angiotensin-converting enzyme inhibitor, has gained increased popularity because it is effective in controlling severe, refractory hypertension. However, there